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Focal inflammation in the brain: role in Alzheimer's disease.

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We hypothesize that amyloid (Abeta) peptide-containing neuritic plaques in the brains of patients with Alzheimer's disease represent chronic inflammatory foci mediated by the actions of the complement system and proinflammatory cytokines. In support of this, in vitro studies show that the (Abeta) peptide is a potent complement activator and that such complement activation leads to the formation of covalent (Abeta)-C3 activation fragment complexes, the generation of the chemokine-like C5a complement activation peptide, and the formation of the proinflammatory C5b-9 complex in functionally active form able to insert into neuronal cell membranes. Other studies show that C5a, together with (Abeta), synergistically augments the release of proinflammatory cytokines from human monocytes. These studies, which provide in vitro support for the hypothesis, are being pursued in an animal model of Alzheimer's disease.

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